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Arsine Intoxication: A Case of Suspected Poisoning in the Steel Industry

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THE JOURNAL OF INDUSTRIAL HYGIENE

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ARSINE INTOXICATION: A CASE OF SUSPECTED POISONING IN THE STEEL INDUSTRY*

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NUMEROUS instances of poisoning by arsine or arseniuretted hydrogen (AsH_3) may be found in the medical literature. Since 1815 a total of 247 cases are on record, of which 50 (or 20 per cent.) had a fatal outcome. The toxic agent in practically all of these cases was produced during the interaction of metals and acids which contained arsenic. Whenever hydrogen gas is generated in a chemical reaction where arsenic is present, a portion of the arsenic will be evolved as arsine.

In the fabrication of sheet steel products, the material is frequently "pickled" in order to cleanse the metal and to remove the surface layer of rust or "mill scale" (Fe_3O_4). The pickling process consists in immersing the steel sheets in a bath of warm, dilute acid for a short time. During the acid treatment, hydrogen gas is evolved. Three instances of arsine poisoning arising from the pickling of iron and steel products have been previously noted. In 1864 Barker (7) cited a mild case of arsine poisoning resulting from the pickling of iron castings in sulphuric acid. Legge (40) later reported seven cases of similar poisoning in the pickling of sheet iron in arsenical hydrochloric acid. A more recent instance of arsine poisoning (76) occurred in pickling steel at the Puget Sound Navy Yard in 1927. Of three cases, one resulted fatally.

At the present time, the pickling of sheet steel constitutes an extensive

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industry. Two factories of one large tin plate mill in the Chicago district pickle a total of 1,900 tons of sheet steel daily. An investigation of the hazard of arsine poisoning in a typical modern pickling operation should therefore be of interest from the standpoint of industrial health. The case of arsenic poisoning which is reported herewith led to a careful survey of the danger of arsine intoxication in this industry.

It should be borne in mind that iron and steel contain small amounts of arsenic as normal impurities, whereas certain other metals, such as tin and particularly zinc, contain larger amounts of arsenic. Zinc is violently attacked by dilute acids with rapid liberation of hydrogen gas. This rapid solution of the metal, together with the copious evolution of hydrogen, creates an ideal situation for the formation of arsine. This possibly accounts for the large proportion of cases of arsine poisoning which have been observed as a result of the action of acids on zinc. The arsenic content of commercial acids also varies considerably. Thus when the sulphur dioxide used in the manufacture of sulphuric acid is obtained from arsenical sulphide ores, the final product is contaminated with large amounts of arsenic. If, on the other hand, the sulphur dioxide is obtained by burning brimstone, which is always remarkably free from arsenic, the resulting acid is practically arsenic free. Since hydrochloric acid is largely manufactured from sulphuric acid and salt, its arsenic content depends upon the amount of arsenic in the sulphuric acid from which it is made.

Case Report

H. B., a laborer, aged 39, was employed in the pickling room of a factory where heavy steel sheets were pressed into beams for automobile frames. His work consisted in piling steel sheets prior to pickling them in hot 5 per cent. sulphuric acid and later, after they had been pickled and washed, in re-piling these sheets. He was employed at this work for a period of nine months and during this interval was apparently in good health except for intermittent attacks of boils resulting from contact with oil of the crude oil bath in which the pickled steel had been dipped. These oil boils may be largely prevented by washing and by cleanly personal habits. He was discharged on account of lack of work and secured new employment as heater in a drop forging factory but worked only a few days before he was again laid off. He then complained of a feeling of tightness across the chest, difficulty in breathing, and general weakness. About two weeks after leaving his work in the pickling room, he suffered an attack of acute gastro-enteritis characterized by vomiting, watery diarrhea, and colicky pains.

A week after this attack he consulted a physician and complained of dyspnea, marked exhaustion, and difficulty in walking. His skin was scaly and had a peculiar bronzed appearance. His condition progressively grew worse and he was taken to a hospital. Tremors and paresthesias of the muscles of the arms and legs developed, followed by paralysis and atrophy of these muscles. There was complete paralysis of the arms and legs. Contractures of the muscles of the hands drew them into claw-like positions. A transverse white band was formed on each of the nails of the hands and feet similar to that described by Aldrich (45) in a case of acute arsenic poisoning. This condition is not characteristic of arsenic intoxication alone, but frequently occurs in typhoid fever and in other diseases associated with severe prostration. At no time during his illness did he report hematuria, jaundice, or pains in the back, although these are the most characteristic and constant symptoms of arsine poisoning.

A twenty-four-hour specimen of urine collected forty-six days after he left his employment in the pickling room was found to contain 0.77 mg. of arsenic, while a similar specimen taken 186 days after he left the pickling work contained 0.023 mg. of arsenic.

About five months after the onset of the neuritis, the skin pigmentation had disappeared; a year later the muscular atrophy, paralysis, and contractures had begun to subside. In order to determine whether or not this condition could have resulted from arsine poisoning incidental to the pickling operation, a careful toxicologic study of the process was made.

Pickling Process

Rolled steel sheets, prior to pressing, are cleaned from a coating of mill scale by pickling in 5 per cent. sulphuric acid heated to 180°F. The steel is packed in piles weighing about 5 tons, in which the individual sheets are separated from each other by the insertion of heavy wires of acid resisting alloy. This permits the pickling acid to act on all surfaces of each sheet. Chains of acid resisting alloy are slipped around these piles of steel and by means of an overhead crane the entire bundle is deposited in the hot pickling bath. Here it remains for about thirty minutes. Then the bundle of sheet steel is again picked up by the crane, dipped into a tank of running water, then into one containing dilute caustic soda solution, and finally into a vat of crude oil. It is then deposited on a large rack where the oil is permitted to drain off. Workmen then unpile the sheets and remove the wire separators.

The twelve workmen who carry on the various operations are furnished with wooden-soled rubber boots, rubber aprons, and cotton gloves. Aside from skin eruptions (oil furunculosis) and occasional headaches, there had been no complaint of illness prior to that of H. B. Some of the men had worked in this pickling room for from three to four years. The pickling room had been in operation about twenty years without a single case of arsenical poisoning having occurred.

The two pickling tanks are made of wood and are 27 feet long, 3 feet wide, and 55 inches deep. They are set into pits so that only 24 inches of the tank extends above the floor level. The tanks are fitted with copper coils for low pressure steam heating. A wooden plug at the bottom of each tank permits drainage of its contents into a sewer. A good idea of the arrangement of the pickling room may be obtained from Figure 1.

Samples of the dilute acid in the pickling tanks are removed from time to time and the amount of free sulphuric acid present is determined by titration. Additions of concentrated (93 per cent.) sulphuric acid from measuring tanks located outside the pickling room are forced by compressed air through iron pipes into the pickling tank. By this means the concentration of free sulphuric acid in the pickling solution is maintained close to 5 per cent. The ferrous sulphate resulting from the action of the acid on the steel gradually increases in concentration; when it has reached the point where the pickling solution has a specific gravity of 1.29 to 1.3, no further additions of acid are made and the spent pickling solution is drained into the sewer. Under ordinary working conditions, pickling tanks are drained every twenty-four to

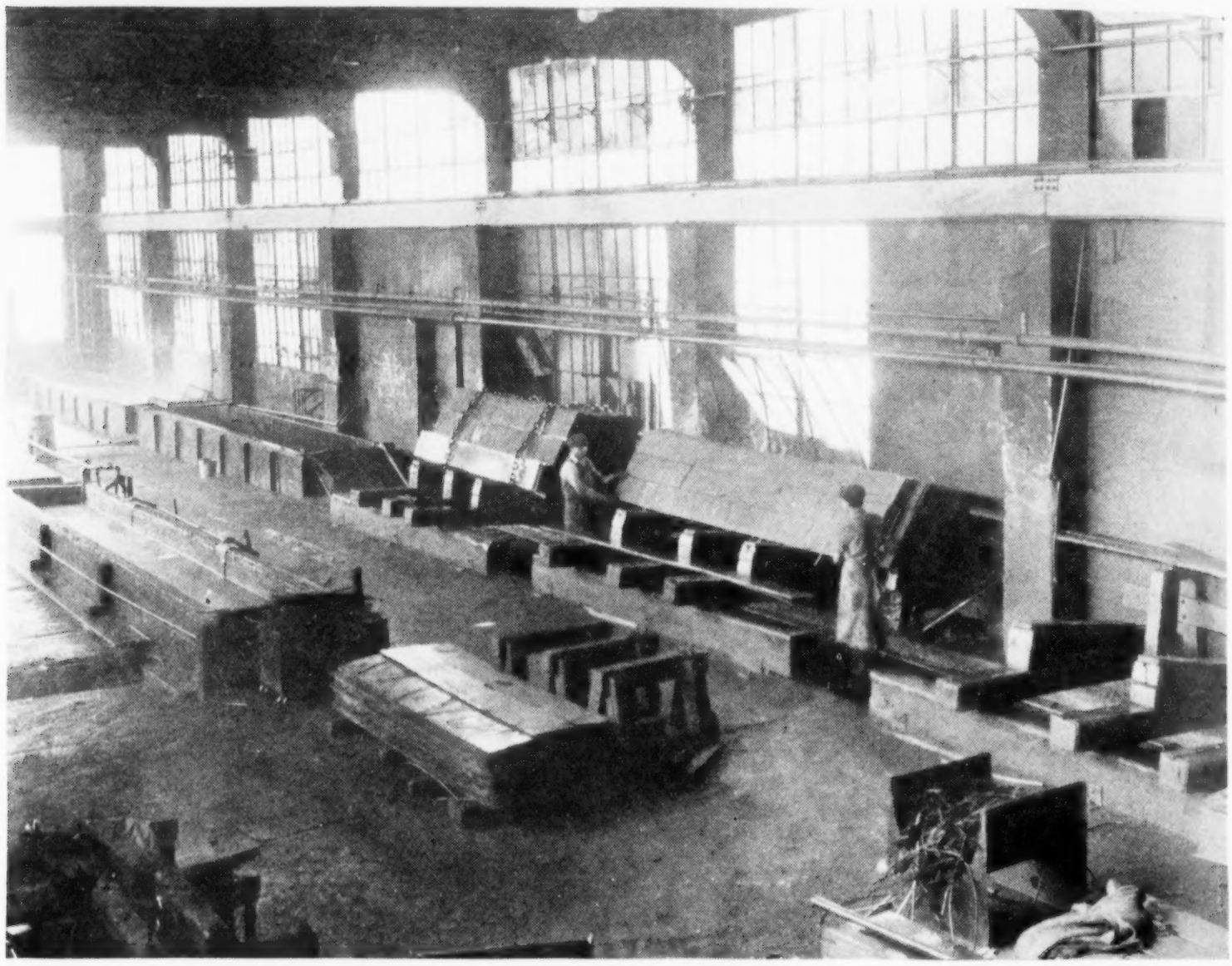

FIG. 1.—The pickling room. The two tanks on the left contain pickling acid. In the left foreground is a pile of sheet steel with wire separators in place. Along the wall in the background are the dilute caustic tank, the crude oil tank, the oil drain rack (with bundle of steel, draining), and the unpiling racks. H. B. worked in the position of the nearest man at the unpiling rack. The tank of wash water is out of the picture in the left background.

thirty-six hours. After draining the spent pickling acid, the hot tank is flushed out with cold water from a hose. A workman then goes into the tank with a shovel and a bucket and removes the sludge which has collected. This residue consists chiefly of mill scale (Fe_3O_4), dirt, rust, and small particles of steel.

During the pickling process, small bubbles of gas arise in the pickling tank as a result of the interaction of the acid and the metal. This gas consists primarily of hydrogen but also contains some hydrogen sulphide, sulphur dioxide, unsaturated hydrocarbons, and a very faint trace of arsine. As the bubbles arise from the tank they produce a fine spray or mist of small particles of liquid. These particles of acid solution produce a sour-smelling fog or haze, which, though not particularly irritating, gives the pickling room its characteristic odor. In order to prevent the carrying of acid liquor into the room by the evolved gases, the pickling solution is covered with a thin layer of either shavings or a patent compound which presumably suppresses the spray formation. During the winter months and in stormy weather, when the win-

dows and roof ventilators in the pickling room may not be opened, forced ventilation at the rate of 55,000 cu. ft. of air per hour per person is supplied by fans. During fair weather, natural ventilation through doors, windows, and skylight ventilators was supplied at a rate of at least 100,000 cu. ft. per hour per man.

A daily average of 390 tons of steel is pickled in this factory.

Chemical Analyses

The sulphuric acid which is used in the pickling process is made by the chamber process from crude sulphur or brimstone. Such acid is known to be of very low arsenic content. Analysis of a typical product showed it to contain 93 per cent. sulphuric acid and 0.2 part per million of arsenic trioxide. When this acid is diluted to 5 per cent. strength, the arsenic concentration of the dilute acid becomes 0.0108 part per million. The steel which was pickled was found to vary in arsenic content between 0.0055 per cent. and 0.011 per cent.

The waste pickling acid at the time it was dumped into the sewer was found to contain from 0.2 to 0.42 part per million of arsenic trioxide. This represents an average increase in arsenic content of thirty times that of the original fresh pickling solution.

An experiment conducted by the late Dr. J. A. Wesener is of great interest in elucidating the relative importance of the arsenic in the acid and of that in the steel, in the formation of arsine during the pickling operation. Dr. Wesener found that on completely dissolving a piece of steel at 188°F. with 5 per cent. arsenic free sulphuric acid, the arsine evolved represented only 2.5 per cent. of the total arsenic contained in the steel. This accounts for the enormous increase in arsenic concentration in the pickling solution between the time it is first used and the time it is dumped into the sewer.

Samples of known volumes of air from various parts of the pickling room were bubbled through a solution of lead acetate to remove hydrogen sulphide and subsequently through dilute silver nitrate solution to absorb any arsine. The silver nitrate solution was then analyzed for its arsenic content by the usual modified Gutzeit procedure.

A sample of 25 liters of air taken over a period of eighty-one minutes at a point 6 inches above the surface of the pickling tank during the pickling of bundles of steel sheets was found to contain 0.006 mg. of arsine.

A sample of 473 liters of air taken at a point about 10 feet from the nearest pickling tank where H. B. worked at unpiling pickled steel sheets was found to contain less than 0.001 mg. of arsine, which is the limit of sensitiveness of the analytic method.

To determine whether or not there was any hazard to the workman who descended into the empty pickling tank to shovel out the sludge, a sample of 27 liters of air was taken at a point 3 feet from the bottom of the empty tank. This was about in the position of the head of the workman while he is shoveling. The shoveling operation required ten minutes and so the sample was taken during this interval. It was found to contain less than 0.001 mg. of arsine.

Mice were exposed for ninety-six hours continuously in the pickling room at the point where H. B. worked

(about 10 feet from the nearest pickling tank). These mice were placed in metabolism cages so that the urine could be collected and examined for hemoglobin. After four days' exposure, there was no evidence of hematuria. These mice were then killed and their bodies, as well as those of the control animals which had been kept in the office of the factory, were each found to contain less than 0.001 mg. of arsenic trioxide when analyzed by the Gutzeit method.

Discussion

In all the previously recorded cases of arsine poisoning resulting from the pickling of iron or steel, the arsenic has been traceable to the acid used. Inasmuch as only a small fraction of the metal is dissolved in the process of pickling and according to Dr. Wesener's experiment only a small percentage of the arsenic in the dissolved portion is volatilized as arsine, the small arsenic content of the steel is not of material importance. Since the arsenic concentration of the pickling acid is of major importance in the formation of arsine, it is obvious that the acid used should be as free from arsenic as practicable. In Legge's cases the acid contained 0.035 per cent. of arsenic trioxide, while in the Puget Sound Navy Yard cases the acid in the pickling tank was found to contain 0.05 per cent. of arsenic trioxide. These represent concentrations of arsenic at least 1,000 times as large as any observed in the pickling tanks tested in this factory.

From the analyses of materials used in the pickling room of the factory in question, it seems highly improbable that the traces of arsine produced could be responsible for the condition of H. B. This conclusion is rendered more certain by the fact that, although he was obviously suffering from the effect of an arsenical intoxication, none of the highly characteristic symptoms of arsine poisoning (hematuria, jaundice, anemia, and pains in the back) were exhibited. The length of time elapsing between cessation of work in the pickling room and the onset of the illness indicates that the traces of arsine in the pickling room could not have been the source of the intoxication since arsine always produces immediate effects.

Arsine very rarely has a violent action on the peripheral nervous system similar to the polyneuritis of H. B. In the cases of arsine poisoning in submarines reported by Dudley (68) there were definite paresthesias, but in only one case (64) has polyneuritis with loss of function been reported following arsine intoxication. The symptoms in this latter case were mild compared with those of H. B.

Unpublished experiments performed under the direction of one of the authors (A. S. L.) have shown that it requires 120 minutes' exposure to a concentration of 0.16 mg. of arsine per liter of air to produce death in dogs. With half of this concentration of arsine, it required twice the length of exposure. If one assumes that the time required to produce death continues to increase in inverse ratio to the arsine concentration, it would require nearly two years' exposure at a concentration of 0.000002 mg. of arsine per liter, which was the amount present at the point where H. B. worked. Such a simple relation of

concentration to time of exposure could not hold for this small amount. When the amount of poison to which a person is exposed becomes sufficiently small, the body is able to withstand its effects for an indefinite period.

The fact that H. B. was still excreting arsenic at the rate of 0.77 mg. of arsenic trioxide per twenty-four hours forty-six days after leaving work in the pickling room and 0.023 mg. 140 days later, indicates that this arsenic was not acquired in the pickling room. Wignall (71) has shown that arsenic is very rapidly excreted in the urine following arsine intoxication. In five cases of acute poisoning he showed that the arsenic concentration of the urine fell from an average of 2.6 mg. of arsenic trioxide per liter immediately after the onset of symptoms, to a concentration of 0.1 mg. of arsenic trioxide per liter (the limit of sensitiveness of his chemical test) thirty-four days later.

With this knowledge of the rapid excretion of arsenic in the urine following arsine intoxication, we can readily see that if H. B. were still excreting 0.77 mg. of arsenic trioxide on the forty-sixth day, the amount which he must have had stored up in his body when he left the pickling room would have represented a ridiculously high tolerance toward arsine. On the other hand, to have been the only one of twelve workmen to have suffered from the traces of arsine in the pickling room, H. B. must have been highly susceptible to arsine. If H. B. received his arsenic in the form of arsine, which is the only way he could have received it incidental to his work in the pickling room, we are forced to assume that he was very hypersusceptible to the action of this substance and at the same time exceedingly tolerant to it. This reduces the proposition to an absurdity.

It seems almost certain that H. B. received arsenic by mouth about the time of the attack of vomiting and diarrhea and that this acute intoxication led to the multiple neuritis.

In conclusion it may be stated that in the pickling of iron and steel products, as now carried on, there is no danger of arsine poisoning so long as the pickling acid used is free from arsenic. The purchase of pickling acid should be made on specifications limiting the arsenic content to 1 part per million, and each shipment of acid should be analyzed for arsenic to guard against arsine intoxication. Records of such analyses should be kept for future reference. Inasmuch as commercial sulphuric acid containing less than 1 part per million of arsenic trioxide is obtainable at very reasonable cost, arsine poisoning in the pickling industry is inexcusable.

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SOME CLINICAL AND PATHOLOGIC OBSERVATIONS ON SILICOSIS IN ONTARIO*

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AND
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DURING the past two and a half years, studies of the pathology produced in the chest by the inhalation of dust, particularly the condition known as silicosis, have been carried on by the Division of Industrial Hygiene of the Ontario Department of Health. In these studies, the Division has had invaluable assistance from the Tuberculosis Division of the same Department.

In the course of these studies 1,487 men have been examined clinically, a stereoscopic X-ray film has been prepared on each man and this film read in conjunction with the clinical examination. Nineteen pairs of lungs obtained at postmortem examination were studied. The percentage of silica, estimated as SiO_2, in the ash of each lung was determined. A certain number of lungs were sectioned and microscopic studies prepared from them. Some of these pathologic specimens were obtained from men who had previously been examined clinically so that it was possible in these cases to link the peculiarities presented at autopsy with the clinical examination.

The men examined were workers from dusty trades, most of whom were exposed to dust containing silica. The few exceptions are included in this report and were studied as controls. The majority of these men were miners. The various occupations represented were mining, quarrying, stonecutting (granite and marble), sandblasting, grindstone operating (sandstone wheels and artificial wheels), smelting, molding, and iron-working (blacksmithing and wrought-iron work). All these operations involve the operator in exposure to dust. Silica is present in all these dusts except that produced in marble cutting, in operating artificial grindstones, and in wrought-iron work. All but these last-mentioned operations were found to produce silicosis.

LEGAL CLASSIFICATION OF CASES OF SILICOSIS

The cases reported below are classified in accordance with the classification in use by the Miners' Phthisis Medical Bureau of South Africa. For the application of this classification we are indebted to Dr. J. M. Smith of that Bureau who visited Canada in the spring of 1927. Dr. Smith personally reviewed the majority of the roentgenograms of this series.

This classification differs from that in general use in the United States for describing the roentgenologic stages of silicosis—*i.e.*, first, second, and third stages as described by Lanza, Pan-

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coast, and others. It is used essentially as a basis for the payment of compensation in connection with the mines on the Rand and has been adopted by the Workmen's Compensation Board in Ontario as the standard for this purpose. Under it, the condition is divided into three legal stages, ante-primary, primary, and secondary. The Act reads as follows:

> A person shall for the purposes of this Act be deemed to have or to have had silicosis,—
>
> (1) In the ante-primary stage, when it is found by the Board that the earliest detectable specific physical signs of silicosis are or have been present, whether or not capacity for work is or has been impaired by such silicosis;
>
> (2) In the primary stage, when it is found by the Board that definite and specific physical signs of silicosis are or have been present, and that capacity for work is or has been impaired by that disease, though not seriously and permanently;
>
> (3) In the secondary stage, when it is found by the Board that definite and specific physical signs of silicosis are or have been present, and that capacity for work is or has been seriously and permanently impaired by that disease or when it is found by the Board that tuberculosis with silicosis is or has been present.

It will be noted that these legal stages provide for diagnosis both during life and at autopsy. Diagnosis during life comprises the results of clinical examination including X-ray. No diagnosis is warranted which is not based on the results of both methods. Roentgenograms of good technical quality are essential.

Ante-Primary Stage.—The X-ray appearances presented by the ante-primary stage are characteristic of the condition. The hila and linear markings are more pronounced. In addition, small rounded nodular shadows can be made out in the lung fields. These appear on the branching shadows cast by the linear markings and between them in what would otherwise correspond to clear lung tissue. This "mottling" is more or less evenly distributed throughout both lung fields. Sometimes there are many nodules present, sometimes few. It has been found that the cut surface of the lung taken from a person whose roentgenogram presents this mottling shows small pigmented nodules in the lung tissue. These nodules vary in size and may be as small as 1 mm. in diameter. They are small areas of fibrosis. It has been arbitrarily decided that this fibrosis must be palpable on the cut lung surface before the condition is considered to be silicosis corresponding to any legal stage of the disease. Fibrosis which can be demonstrated by microscopic sections of the tissue but is not demonstrable in the gross specimens, both by sight and by touch, is not considered sufficiently specific to warrant diagnosis. There appear to be definite "selective areas" where this nodulation appears early and is more marked. Such areas are the inner and middle zones around the lung roots, especially on the right side.

Primary Stage.—In this stage, the roentgenogram presents the same general appearance as in the ante-primary stage. The individual nodular shadows are, however, larger and there are more of them. The nodules appear more in the clear areas and less on the linear shadows. The hila and linear shadows are less in evidence.

Secondary Stage.—The picture presented by the secondary stage of pure

silicosis is an exaggeration of the foregoing. The nodules are still larger, are more densely packed, and correspond obviously to massive fibrous replacement of lung tissue, with the hila and linear markings generally not in evidence. As indicated by the legal definition, silicosis in any stage, definitely complicated by tuberculosis, is classified as secondary. To this category should also be added the cases in which the fibrosis is so widespread and dense that the corresponding shadows in the roentgenogram have not the mottled characteristic. In these cases, it is often difficult to say, except by observation and clinical examination, that tuberculosis is not present. This point can be settled postmortem, and frequently only then.

The foregoing description refers to typical silicosis. Certain atypical cases are frequently encountered. One is forced to this conclusion as a result of autopsy findings and roentgenograms. In certain instances, the result at autopsy has been considered in connection with clinical findings. In some cases in which tuberculosis cannot be demonstrated, the mottling generally seen in the roentgenogram is not in particular evidence but seems to be replaced by a general hazy density appearing in the situations usually associated with mottling.

It should always be kept in mind that the legal classification referred to is based not only on X-ray diagnosis but also on clinical signs and symptoms. Thus in placing a case in the proper category both are considered. For example, a case in which the roentgenogram indicates the early stage but in which there is disablement and poor physical condition with no other cause than silicosis to account for the disablement would not be considered ante-primary but either primary or secondary as the condition warranted. Where silicosis is present and there is also definite tuberculosis, the case is always secondary.

Among the 1,487 workers examined in the course of this survey fifty-three had silicosis in the ante-primary stage, sixteen in the primary, and eleven in the secondary. In addition, thirty-seven persons in whom silicosis could not be demonstrated were found to be suffering from tuberculosis.

Dust Exposure and Dust in Lungs

The dusts to which the foregoing workers were exposed were from various operations and contained various amounts of silica. The dusts were not subject to analysis but in most cases the substance which was responsible for the dust was analyzed as to its silica content; this varied from 2 to 99 per cent., estimated as SiO_2. The lowest amounts that were found in this survey to be involved in the production of silicosis were from 10 to 14 per cent.; the highest, 99 per cent. Where conditions were comparable—*i.e.*, among the miners—the number of cases discovered varied with the silica content of the materials (rock) worked.

Estimation of Silica in Lungs

Estimation was made of the percentage of silica in lungs obtained at autopsies from workers, some of whom were employed in occupations involving exposure to silica dust and some in occupations where there was either no exposure or where the amount of

silica was small. Thus nineteen pairs of lungs were examined—fourteen from persons who were exposed to silica dusts in their occupations, and five from those whose work did not involve such exposure. The method of analysis was as follows:

From 50 to 450 gm. of wet lung (dried with a towel after removal from formalin solution) were weighed

TABLE 1.—RESULTS OF ANALYSES OF LUNGS FOR SILICA CONTENT

LAB. NO.	DATE	NAME	SILICA IN ASH OF DRIED LUNG	REMARKS
			%	
124	Oct. 22, 1924	L. H.	33.26	Worked in crushing room in quartz quarry for 4 seasons of 6 months each. Died of silicosis.
132	Nov. 12, 1924	T. G. H.	1.28	City dweller. Normal.
133	Nov. 12, 1924	T. G. H.	2.67	City dweller. Normal.
130	Nov. 2, 1925	K.	39.80	Driller for 7 years. Died of silicosis.
131	Nov. 3, 1925	Sand blaster	16.83	Sand blaster for 15 years. Died of silicosis.
323	Jan. 27, 1926	F. L.	15.29	Drilling, etc., for 17 years. Died of silicosis and tuberculosis.
529	Aug. 26, 1926	B. P.	0.77	Stone worker (marble, etc.). Killed.
753	Oct. 5, 1926	F. C.	18.86	Drilling and blasting for 7½ years. Moderate silica exposure. Killed.
985	Dec. 23, 1926	C. D.	33.20	Driller for 16 years. Died of silicosis.
1,017	Apr. 4, 1927	W. G.	2.34	Marble cutter for 20 years. Died of tuberculosis.
1,022	May 15, 1927	J. K.	7.60	Mucker for over 3 years. Killed.
1,034	Oct. 15, 1927	A. B. #1499 H	28.86	Driller underground for 13 years. Fracture of base of skull.
1,035	Oct. 15, 1927	M. V. #1676 H	7.40	Driller for 8 years in mines of low silica content. Died of myocarditis.
1,036	Oct. 15, 1927	E. C. #1100 H	26.02	Driller underground for 8 years. Fractured skull.
1,037	Oct. 15, 1927	J. F. #2701 H	7.82	Slight silica exposure for 6 years. Killed.
1,038	Oct. 15, 1927	R. B. #2874 H	36.89	Driller for 7 years. Killed.
1,039	Oct. 15, 1927	M. K. #17 McI	27.32	Driller for 13 years. Killed.
1,040	Oct. 15, 1927	S. W.	15.23	Mining for 9 years underground. Died of silicosis complicated with tuberculosis.
1,069	Nov. 24, 1927	T. G. H. A-272–27	0.60	City dweller. Died of pneumonia.

into large porcelain evaporating dishes and dried in an electric oven at 100 to 107°C. for forty-eight hours. The weight of dried lung was observed and the sample was then transferred to a tared platinum dish and carefully ignited. The ignition was completed in a muffle. The dish and contents were weighed and the amount of ash was noted. If the ash was small, the whole portion was fused with sodium carbonate. If the weight of the ash exceeded 3 gm., it was mixed up and a 1-gm. portion fused with sodium carbonate in a platinum crucible. (In some instances, a fusion mixture, sodium and potassium carbonate, was used. It was thought best to use sodium carbonate alone as some silicates require a higher fusion temperature than that obtained with the fusion mixture.) The contents of the crucible were then transferred to a 250-c.c. pyrex beaker and the melt dissolved with hot distilled water followed by hydrochloric acid. The entire volume was then taken down to dryness and baked at 120° to 130°C. overnight in an electric oven. Concentrated hydrochloric acid was added, then water, and the solution heated to boiling. The white residue was filtered off, washed with hot distilled water, and dried for two hours at 105°C. Ignition in a platinum crucible followed and the crucible and contents were weighed. A few drops of concentrated sulphuric acid and 10 to 15 c.c. of hydrofluoric acid were added. The crucible was heated at first over a low flame and after volatilization of the acids over a Méker burner. It was then cooled and weighed, and the percentage of SiO_2 recorded.

The results presented in Table 1 are from estimations of selected areas of the lungs and in general represent the silica content of the more fibrosed areas.

Discussion

The lungs previously referred to have afforded an opportunity to study the pathology of the condition as well as yielding material for chemical analysis. Some of the specimens were obtained from men who had been previously examined and through them it has been possible to link the clinical and the X-ray findings with the pathology as presented.

It is not the purpose of this paper to do other than discuss several interesting points that have come out of these studies and to endeavor to illustrate them by reference to specific cases. The general pathology of the condition has been amply described many times before this.

No definite case of either anteprimary or primary silicosis has been studied pathologically by us. Such cases come to autopsy only as the result of accident or as the result of death from some cause other than silicosis. The material to hand includes, however, one specimen (the microphotograph in Fig. 1) from a man previously examined, whose condition was approaching the earlier stage.

The roentgenogram in this case showed hila which were dense and widened. The linear markings were prominent, especially those corresponding to the descending bronchi, and were beaded along their courses particularly at the points of branching. The lungs here showed some thicken-

ing of the pleura with a few adhesion tags. They were air holding throughout and floated in water. The tracheobronchial lymph glands were dark in color, and firm. Some were fibrosed. The lung cut with considerable resistance and the cut surface showed much emphysema and many pigmented areas of mottling. These pigmented areas were not palpable. Microscopic section showed besides normal lung tissue many areas where the interstitial tissue of the alveolar walls was increased and also much general fibrosis following the lymphatics. Throughout the section were seen many pigment gran-

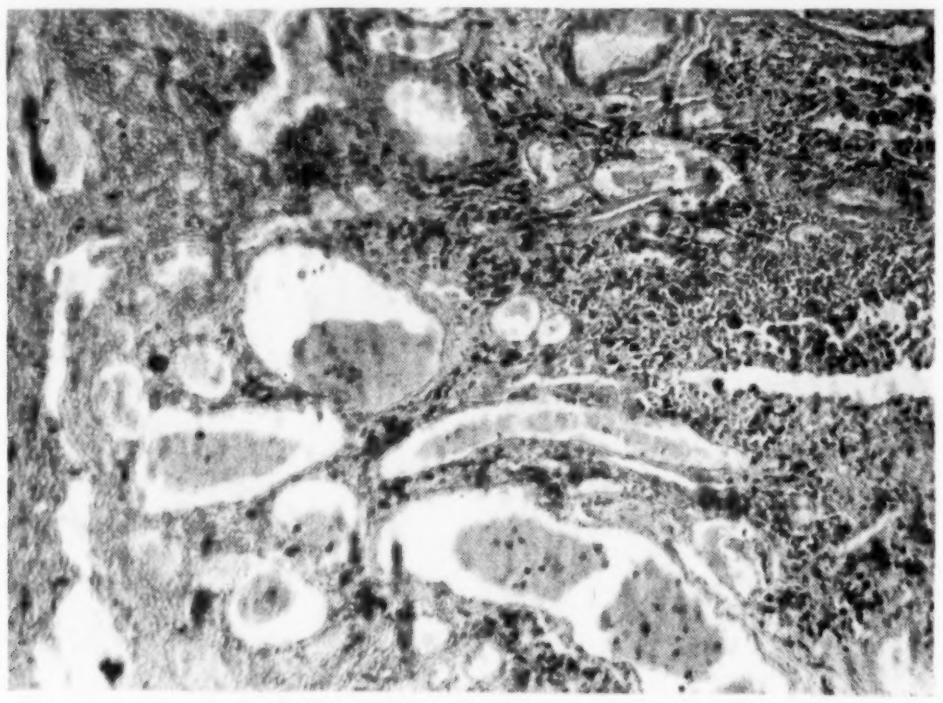


FIG. 1.—Note the aggregation of pigment about the vessels; the pigment filled cells in and about the vessels and alveoli; the invasion of the alveolar septums with fibrous tissue; and the collection of blood cells in the alveoli among which are seen the pigment holding cells.

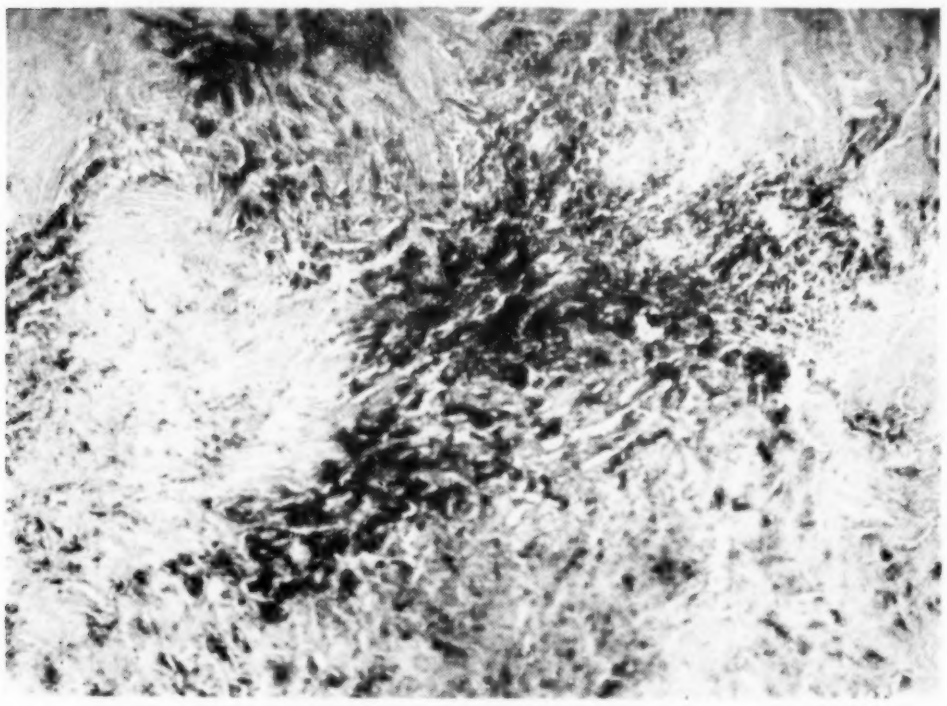


FIG. 2.—Note the extensive growth of fibrous tissue tending to grow in whorls; also the heavy deposition of pigment particles.

ules. Much of this pigment was contained in endothelial cells. The silicotic content of this lung is given under No. 753 in Table 1.

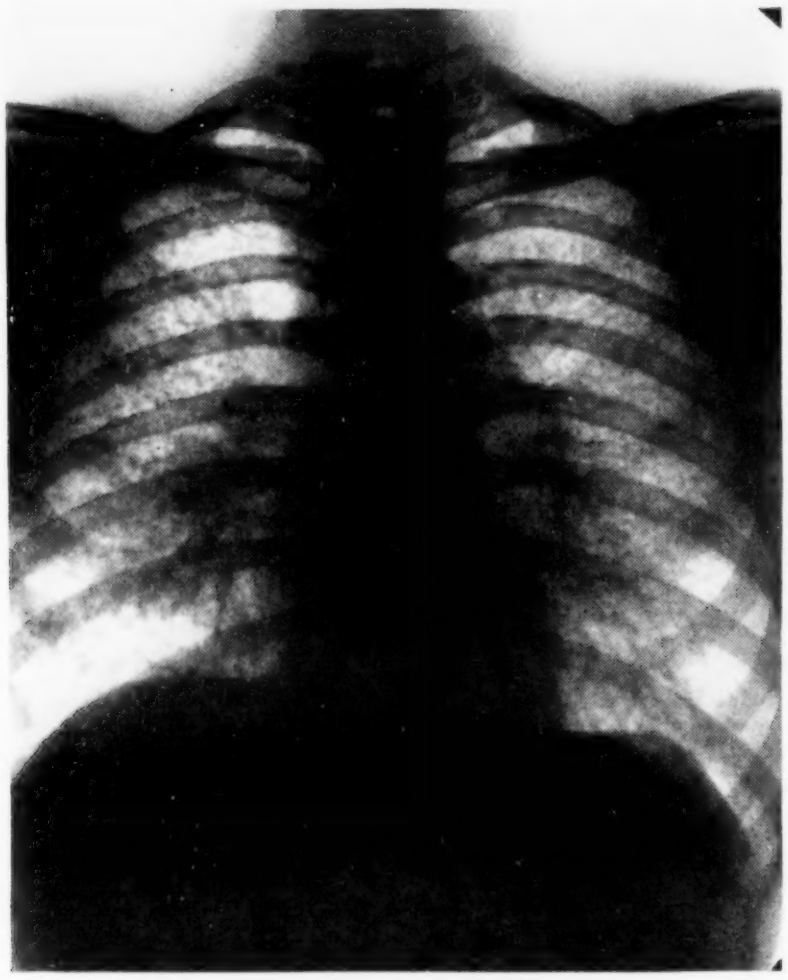
Figure 2 is a microphotograph of a section of lung from a case which it is felt may illustrate those previously referred to as atypical. Unfortunately no examination was made before death in this case. The man from whom these lungs were obtained died of uncomplicated silicosis. No other cause of death could be demonstrated. These lungs were larger and heavier than normal. The pleura was thickened and showed adhesion tags. The lungs sank when placed in water although they appeared air holding throughout. They cut with very marked resistance and grating of the knife. The tracheobronchial glands were thick and fibrosed. The cut surface showed much pigment, emphysema, and innumerable, minute, dark fibrosed areas throughout, especially in the middle and lower thirds. No definite nodules could be palpated but the whole tissue felt firm and resistant as though combined with rubber. Close inspection showed the alveoli dilated and surrounded by dark, dense tissue.

The microscopic section showed massive replacement of normal lung tissue by fibrous tissue containing much pigment. The alveoli were dilated, their walls were thickened, and in some cases were fibrosed. The alveoli contained endothelial cells filled with pigment. Many "heart failure" cells were seen throughout. The interstitial tissue contained young blood vessels surrounded by endothelial cells filled with pigment. The blood vessels were sclerosed. The amount of fibrous tissue and pigment was greater in the neighborhood of the blood vessels. The chemical findings are given in Table 1, under No. 985.

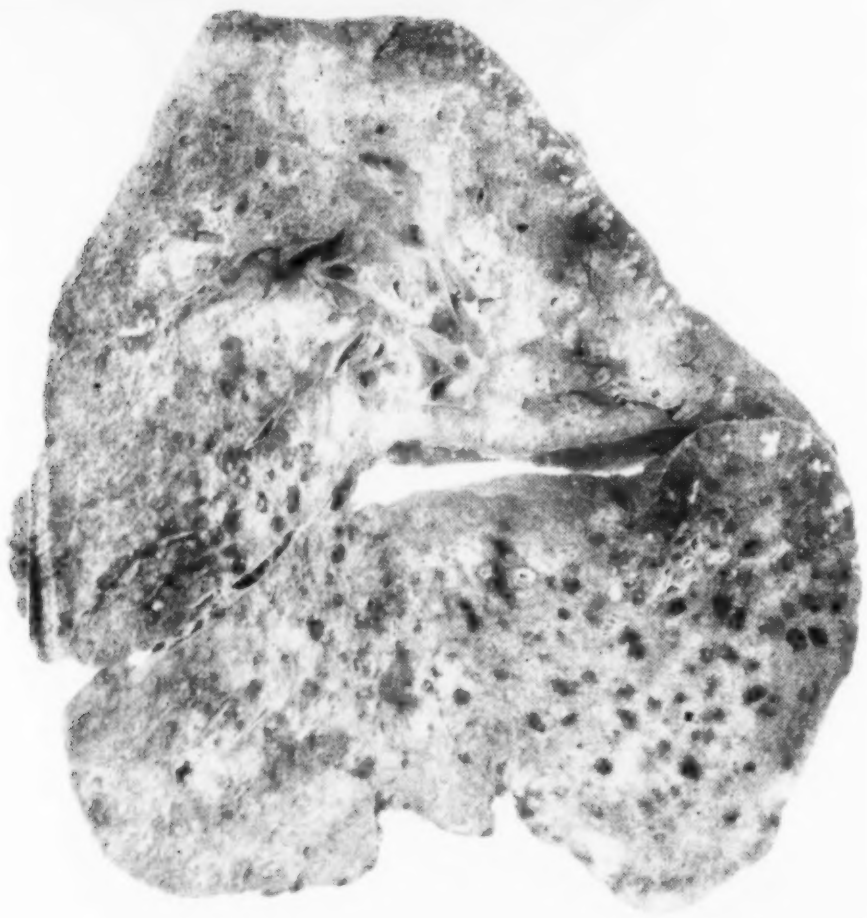
Figures 3, 4, and 5 illustrate series from examined cases. They are included to show the X-ray appearance and the corresponding pathology.

Figure 3 is interesting. This is from a secondary case. The man died of silicosis complicated by tuberculosis. The roentgenogram is a good example of the appearance of primary silicosis. The mottling is moderately large and widespread. The cut of the gross specimen illustrates the corresponding mottled areas in the lung tissue. These areas were densely fibrosed and palpable. The light-colored nodules are tuberculous foci; their position can be noted and also the emphysema showing throughout the specimen. The pleura in this case was thickened, the lungs appeared normal in size, they were slightly heavy but floated in water. They cut with resistance and grating. Microscopic section (see Fig. 3) showed some areas of almost normal lung tissue. The alveoli in these areas contained large phagocytic cells filled with fine pigment granules. Interspaced with these areas were others in which the normal structure was obliterated by dense fibrosis. The fibrous tissue was seen occupying the alveolar walls where such could be identified. It could be seen in some places budding out into alveoli, in others surrounding alveoli indicating how obliteration would eventually be accomplished. The fibrosis and pigmentation were also marked in the neighborhood of blood vessels and bronchi.

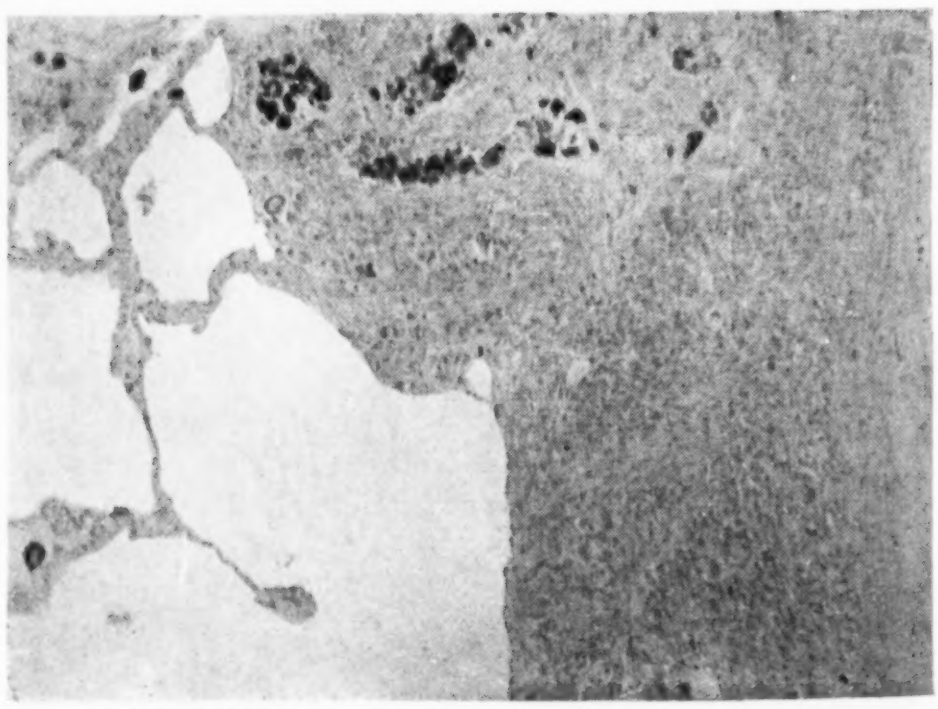
No. 1,040 in Table 1 indicates the percentage of silica in the ash of this lung.



Patient died of silicosis complicated with tuberculosis. Note the medium and coarse mottling distributed throughout both lung fields.



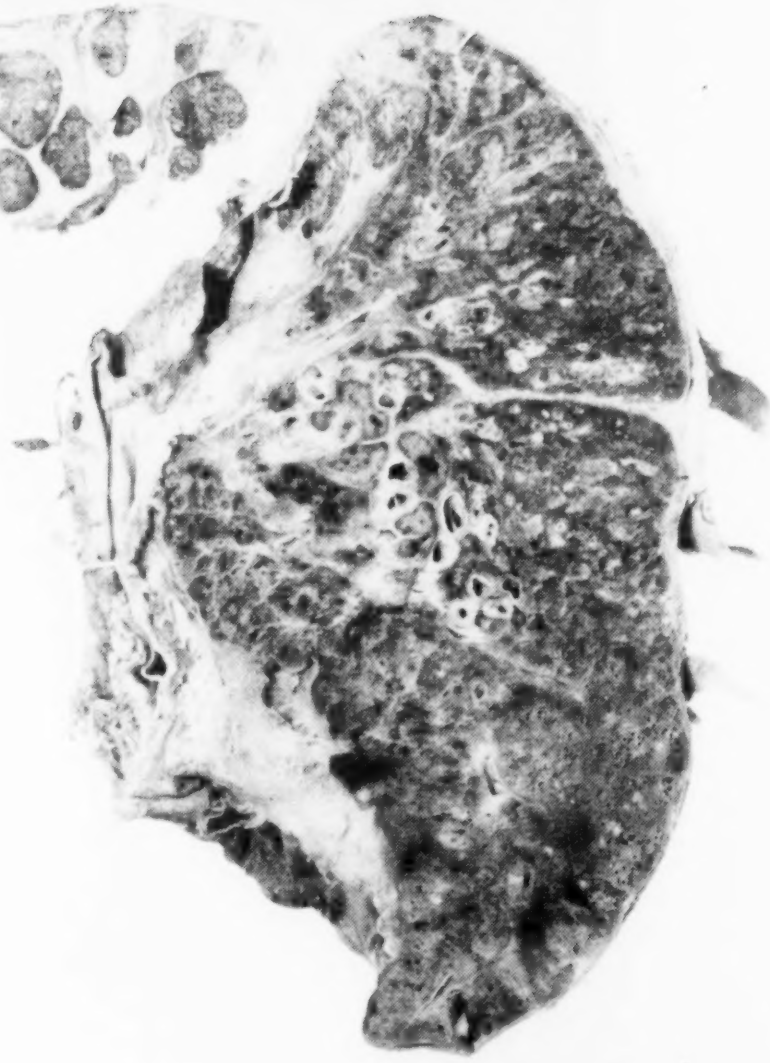
Note the emphysema; the pigmented nodules scattered throughout; and the tuberculous foci, especially beneath the pleura.



Note the fibrous tissue invading the alveolar septums; the pigment filled dust cells; the massive fibrosis which has obliterated the normal lung structure. This section was made through the edge of a nodule.

FIGURE 3

Patient died of uncomplicated silicosis; Note the dense shadows in the upper two-thirds of the lung fields; and the distortion and peaking of the diaphragm on the right side.



Note the corresponding dense fibrosis; the thickened pleura; adhesion tags; emphysema; and fibrosed bronchial glands.

Note the massive fibrosis tending to grow in whorl formation; the pigment and particles scattered throughout the section. Much of the pigment is collected in the neighborhood of the blood vessels.

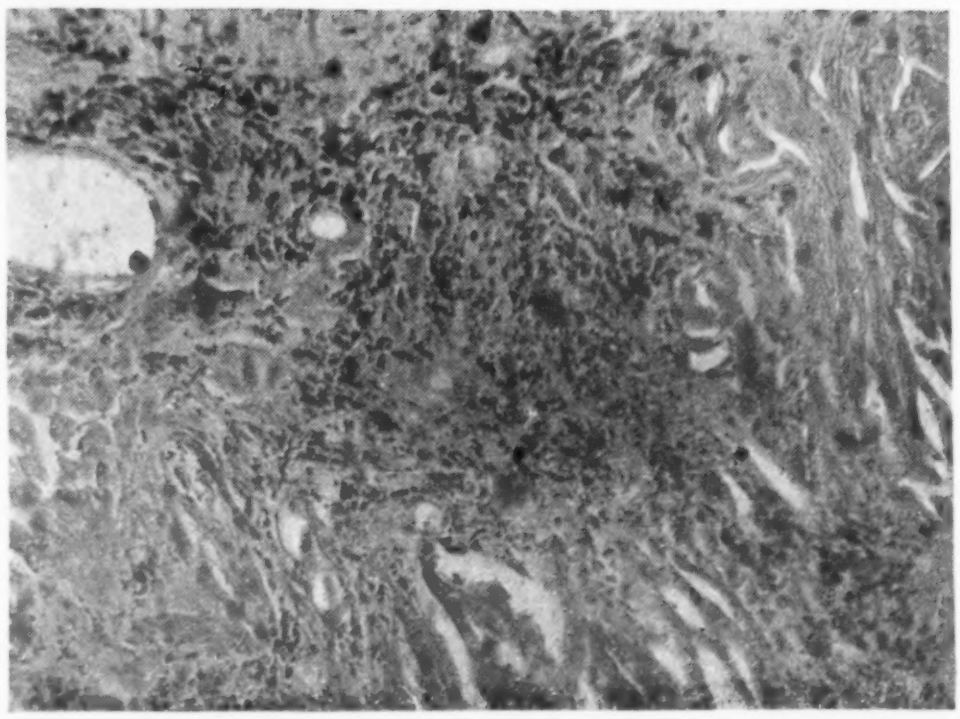
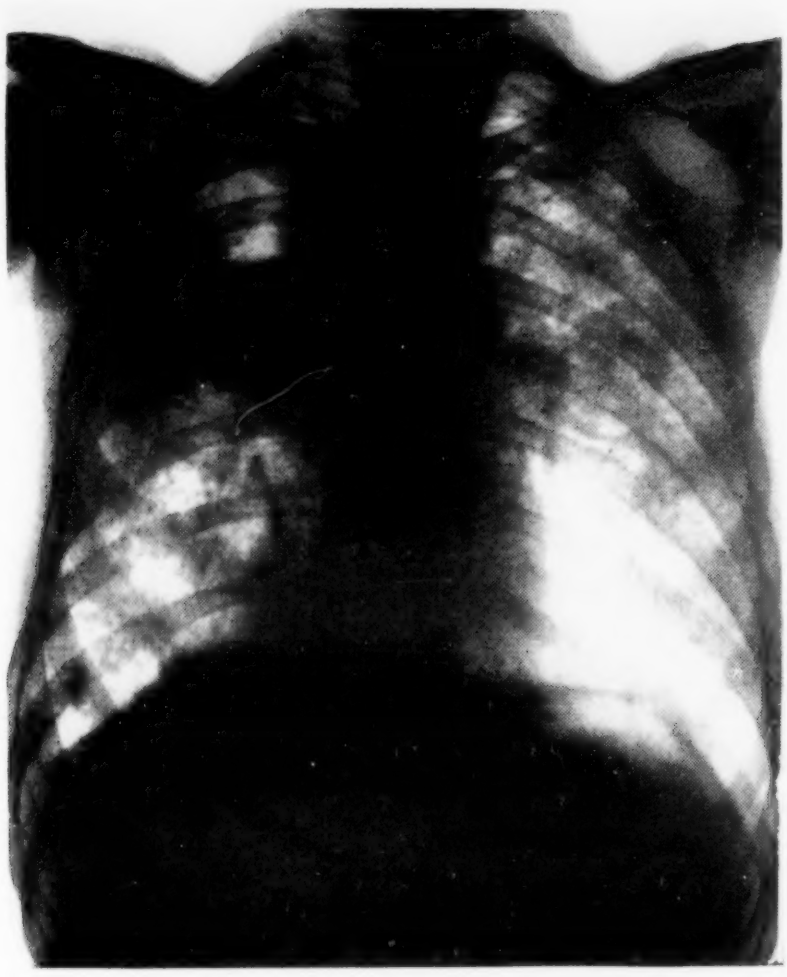
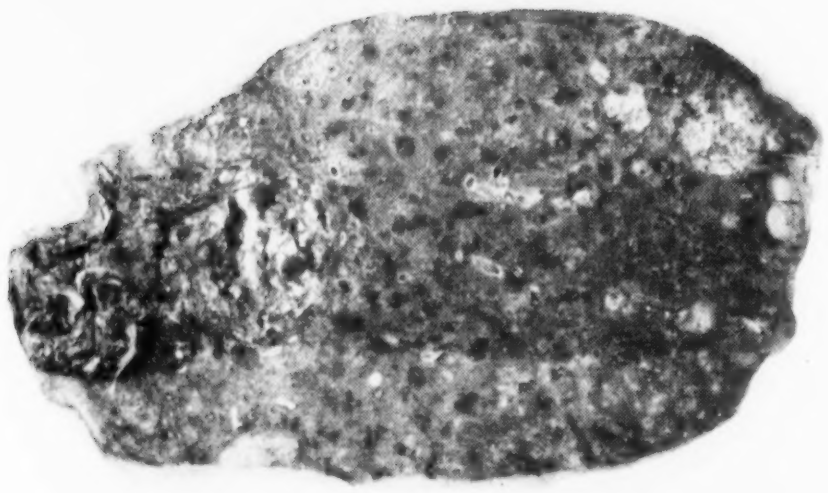


FIGURE 4



Case of silicosis complicated with tuberculosis (six weeks before death). Note the area of fibrosis in the right apex, with cavity formation; the long, narrow heart shadow; the diffuse mottling throughout the remainder of the lung fields.



Note the pigmented nodules of fibrosis; also the tuberculous foci and cavities.

Note the invasion of the lung tissue by fibrous tissue; also the pigment particles scattered throughout, with the aggregation about the blood vessels.

FIGURE 5

Figure 4 was produced from the roentgenogram and specimen of our first case of silicosis. This illustrates the massive type of fibrosis. The connection between the X-ray appearance and that of the pathologic specimen can, it is felt, be readily appreciated.

The lungs in this case bulged out of the chest when the thorax was opened; the pleura was very much thickened and was strongly bound to the parietal pleura with adhesions. The lung sank in water. It cut with much resistance and grating. The majority of the lung was non-air holding. The tracheobronchial glands were thick and fibrosed. The microscopic sections show scarcely any areas of lung tissue approaching normal. The fibrous tissue was massive. Much pigment was present throughout, but chiefly around the vascular channels which showed sclerosis of the vessel walls. No tuberculosis could be demonstrated in this case.

The silica content of this lung can be seen by reference to No. 124 in Table 1.

Figure 5 illustrates a combination of types 1 and 2. In this case, there was widespread involvement by tuberculosis with cavitation. The massively fibrosed areas were almost completely dissolved by this process. The lungs were bound to the parietal pleura. Both upper thirds of the lungs were occupied by large cavities surrounded by extremely dense fibrous tissue. Portions of the lung floated but showed nodular fibrosed areas with nodules palpable. The roentgenogram in this case was made at the time of examination six weeks before death. The cavity formation then present can be noted. The corresponding area of the opposite side as previously remarked was also found extensively cavitied at autopsy.

The silica content of the lungs in this case is indicated under No. 323.

BOOK REVIEWS

WORKERS' HEALTH AND SAFETY: A STATISTICAL PROGRAM. By *Robert Morse Woodbury*, with the aid of the Council and Staff of the Institute of Economics. Cloth. Pp. 207 with index. New York: The Macmillan Co., 1927.

This book, which covers a very wide field, is a fair statement of many things that ought to be done or that are being accomplished inadequately at the present time. It will prove disappointing to those in medical control of industry in that it contains little indication of how to do the various things cited as imminently important. For example, in the summary we find:

> For health protection, six types of statistics are required. (1) To measure the prevalence of physical defects, data based upon physical examinations of workers and analyzed by occupation and industry are needed. (2) To measure occupational morbidity, statistics of sickness should be available, based either upon records of absences from work due to sickness or upon records of sickness kept in connection with benefit funds or health insurance; and the number of cases of sickness should be related to the number of cases exposed to risk. These data should be analyzed not only by causes of sickness, but also by sex, age, industry, and occupation. Special statistics on the prevalence of specific occupational disease, so-called, should be collected. (3) To show the responsibility of occupation over disease and death, further, statistics of occupational mortality are required. For sound conclusions the deaths in each occupation should be compared with the population at risk and analyzed by sex and age. (4) To show the prevalence of health hazards, statistics are needed on the extent to which materials or processes, for example, the spray-coating process, known to involve health hazards are used. (5) On the subject of methods and policies of health protection data are required, for example, to show the prevalence of health examinations in industry and of industrial clinics, the extent to which preventive treatment is made available to workers in industry, and the value of each such method of health protection. Finally, (6) to test the results and progress of prevention, comprehensive records of sickness and mortality, compiled in a uniform manner from year to year or over 10-year periods, are required; they should throw light upon changes in prevalence of defects, in incidence of sickness, and in the trend of occupational mortality rates.

Those now in charge of health departments in our large industries will agree readily enough that such items of information would prove interesting and profitable. Indeed, the more thoughtful industrial physicians have known all these necessities for a long time and their need is not for lists of what should be done but for constructive suggestions as to how to do it. The technic of gathering reliable morbidity figures in industry is both difficult and expensive. In the neighborhood of Boston I know of but two firms which are making a successful start at such a task.

Doubtless the book will have utility as a "program" of things that should be done and as a critique of what has been done but badly in the past, but as a constructive basis for actual work industrial physicians will not find it impressive.—*C. K. Drinker.*

THE MANUAL OF INDUSTRIAL SAFETY. By *Sidney J. Williams*, C.E., Director, Public Safety Division, National Safety Council; Formerly, Chief Engineer, National Safety Council; Chief Engineer, Wisconsin Industrial Commission. Cloth. Pp. 197 with appendixes and index. Chicago and New York: A. W. Shaw Co., 1927.

The author presents the subject of industrial safety from the administrative standpoint. The major portion of the book deals with safety organization and methods, rather than with problems of mechanical safeguarding.

"The basis of the safety man's job is this fact . . . . : that the men and women who work for a manufacturing company, a mine, a railroad, or other employer are the most valuable assets the company has and that their safety, comfort, welfare, and good will are essential to the company's success." Unless the employer believes enthusiastically in safety work, the safety man will be unable to accomplish much.

Problems of industrial safety involve engineering skill, but, to a greater degree, organization and education of the staff and employees. The safety man cannot carry on his job alone. He must have the close co-operation of his associates. He must be diplomatic. Above all things, he must secure the co-operation of the departmental foremen. The men look to their foremen for leadership and the attitude of the foremen toward safety work will be largely reflected in the attitude of the men. By acting as a friendly adviser to the foreman and recognizing his authority, the safety man can do much toward gaining co-operation.

In the first part of the book the author discusses the many problems of administration. The procedure to follow in organizing a safety campaign and the value and organization of safety committees are discussed. Several chapters are devoted to safety meetings, publicity work, and competitions.

In the chapter devoted to safety inspections a valuable general check list of things to look for is included. In addition to this general list, special lists for specific industries are given in an appendix.

The fundamentals of accident recording, accident statistics, and methods of computing accident rates are given briefly in one chapter. It is perhaps unfortunate that more space is not given to this important phase of safety administration.

The fundamental principles of mechanical safeguarding are treated in the second part of the book. No attempt is made to give detailed information on construction of safeguards or on methods of protection for specific industries or machines. The scope of the book does not permit it. A valuable chapter in this section deals with accident prevention through proper planning and construction of the factory. The value of early consultation before construction is started is pointed out.—*Theodore Hatch.*

BOOKS RECEIVED

Books received are acknowledged in this column, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers and as space permits.

INTERNATIONAL CLINICS. Volume IV. Thirty-Seventh Series, 1927. Cloth. Pp. 309 with index. Philadelphia and London: J. B. Lippincott Co., 1927.

CONVALESCENCE, HISTORICAL AND PRACTICAL. By *John Bryant*, M.D. Cloth. Pp. xvi, 269 with index; illustrated. New York: The Sturgis Fund of the Burke Foundation, 1927.

HANDBOOK ON DIET. By *Eugene E. Marcovici*, M.D., Formerly Assistant to Professor von Noorden in Vienna; Instructor, Post-Graduate Hospital; Assistant Attending Physician, Roosevelt Hospital, Out-Patient Department, New York. Cloth. Pp. viii, 323 with index. Philadelphia: F. A. Davis Co., 1928.

WHAT YOU SHOULD KNOW ABOUT HEALTH AND DISEASE. By *Howard W. Haggard*, Associate Professor of Applied Physiology, Yale University. With an introduction by Yandell Henderson. Cloth. Pp. xiii, 538 with index and illustrations. New York and London: Harper & Brothers, 1928.

DIABETES: ITS TREATMENT BY INSULIN AND DIET. A HANDBOOK FOR THE PATIENT. By *Orlando H. Petty*, A.M., M.D., F.A.C.P., Professor of Diseases of Metabolism, Graduate School of Medicine, University of Pennsylvania; Physician in Charge of Departments of Diseases of Metabolism, Hospitals of the Graduate School of Medicine, University of Pennsylvania, and Philadelphia General Hospital; Consultant in Diseases of Nutrition and Metabolism, Shriners' Hospitals for Crippled Children, Philadelphia Unit. Fourth revised and enlarged edition. Cloth. Pp. 155 with index. Philadelphia: F. A. Davis Co., 1928.

MECHANICS AND CHEMISTRY OF THE HUMAN BODY. (A Sequel to "Colonic Therapy.") By *O. Boto Schellberg*, New York City. Cloth. Pp. 44 and illustrations. New York: Schellberg Institute, Inc., 1928.

Fifth International Medical Congress for Industrial Accidents and Occupational Diseases

Budapest, September 2 to 8, 1928

The Executive Committee of this Congress consists of the following: President, Dr. Tibor de Verebély, Professor at the University; Vice-President, Dr. William de Friedrich, Professor at the University; Secretary-General, Privatdozent Dr. George Gortvay, Section Chief.

The National Committee for the United States has been created and consists of the following: Dr. Volney S. Cheney, Chicago; Dr. R. W. Corwin, Pueblo; Dr. Eugene L. Fisk, New York; Dr. Otto P. Geier, Cincinnati; Dr. Leonard Greenburg, New Haven; Dr. George M. Kober, Washington, D. C.; Dr. W. J. McConnell, Philadelphia; Dr. Lloyd Noland, Birmingham; Dr. Francis D. Patterson, Philadelphia; Dr. George M. Price, New York; Dr. Frank L. Rector, Chicago; Dr. Wm. A. Sawyer, Rochester; Dr. Henry F. Smyth, Philadelphia; Dr. C.-E. A. Winslow, New Haven; and Dr. Emery R. Hayhurst, Columbus, *Chairman*.

Addresses already scheduled by various prominent Europeans include the following: Prof. J. Liniger, Frankfurt a. M.; Dr. F. Zollinger, Aarau; Prof. K. B. Lehmann, Würzburg; Sir Thomas Oliver, London; Prof. J. G. Sleeswijk, Delft; Prof. Jötten, Münster; Prof. Koelsch, München; Prof. Julius van der Hoeve, Leiden; Prof. Dr. Stephan Jellinek, Wien. Also lectures are scheduled

to date by the following: Prof. Salvatore Diez, Roma; Dr. C. Poenaru Caplescu and Dr. Presbeanu, Bukárest; Prof. Theodor Sommerfeld, Berlin; Dr. Lorenz Böhler, Wien; Prof. Dr. Quensel, Leipzig; Prof. Dr. C. Marcus, Breslau; Prof. Dr. Molineus, Düsseldorf; and Sanitätsrat Dr. Alfred Peyser, Berlin-Charlottenburg.

Addresses and lectures are wanted from American physicians, dentists, and other specialists in the field. Such are requested to get in touch with the Chairman for the National Committee for the United States, Dr. Emery R. Hayhurst, Hamilton Hall, Ohio State University, Columbus, Ohio. General invitation is also extended to attend the Congress which will be arranged so as to coordinate with the Deutscher Naturforscher Tag to be held in Hamburg, and the Orthopädenkongress to be held at Prague during the month of September, 1928.

Fourth International Session of the Permanent International Committee on Occupational Diseases

Lyons, April 3 to 6, 1929

The questions on the agenda of the Session are as follows:

1. Silicosis (three reports: etiology, clinics, and legislation); a general report dealing with forms of pneumonokoniosis.
2. Cataract of occupational origin (two reports: etiology and clinics).
3. Endocrine system and forms of poisoning (it is intended to furnish under this heading a single report to be intrusted to three experts).
4. Communications relating to results of inquiries so far unpublished.

Finally, if it is found advisable to include another question on the agenda, the subject chosen will be the problem of occupational disease statistics.

Prof. Etienne Martin, of the Institute of Legal Medicine of Lyons, in collaboration with Professors Agasse-Lafont and Kohn-Abrest of Paris, with the approval of the executive will designate the experts selected to present reports on the subjects before the Session.

Requests for all further information should be addressed to Monsieur le Prof. Etienne Martin, or to the Secretariat of the Committee, Hygiene Service, International Labour Office, Geneva.

List of Members

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South Africa	Prof. Mavrogordato
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	Dr. Badham (Sydney)
Japan	Dr. Kose (Tokyo)
	Prof. Teruoka (Kurashiki) (Okayama)

Convention of National Nursing Organizations

Louisville, June 4 to 9, 1928

Industrial Nursing will receive its share of attention at the meetings held by the Industrial Nursing Section of the National Organization of Public Health Nurses. "Ethics in Industrial Nursing" will be the topic at the luncheon of that section on Thursday, June 7, at the Brown Hotel; and at the meeting which will precede the luncheon the subjects will be "Nutrition Work in Industry" and "Adequate Preliminary Training for Industrial Nursing."

Information regarding details of the convention can be obtained from the American Nurses' Association, 370 Seventh Avenue, New York City.

J. I. H.
May, 1928